Pediatrician's Point of View

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ALTHOUGH THE SUBJECT is hyaline membrane disease, I prefer the epithet hyaline membrane atelectasis, for it is atelectasis that actually causes the trouble; the hyaline membrane happens to be the disturbing feature in the causation. Atelectasis of the newborn has long been recognized as a very serious threat to the survival of newborn infants, particularly those born prematurely. This term used to be used, and often without qualification, on death certificates as an adequate diagnosis, and is still so used by some physicians. This practice was not seriously challenged until 1933, when Farber and Wilson[1] published the results of a study of atelectasis of the newborn in which they called attention to the many and varied causes of atelectasis and made the statement that a diagnosis of atelectasis was not a diagnosis at all without a qualifying statement as to etiology. I think that more and more of us are finding that this statement is true. There are some cases of atelectasis in the premature that have practically no cause excepting prematurity. That is a little different, because the lungs in those very small prematures are uninflatable. Farber and Wilson also described a condition which I think now we would call hyaline membrane. They discussed obstruction to the flow of air to and from the lungs caused by aspiration of amniotic fluid, making particular mention of the vernix in this aspirated material in the bronchi, and noting that squamous cells were present in the lungs of term infants but not in the lungs of infants born quite prematurely. The material that they called vernix (also known as asphyxial membrane) is what is now called hyaline membrane. By asphyxial membrane they meant that this material, plastered against the wall of the alveoli, alveolar ducts and respiratory bronchioles, interfered with the exchange of gases between the capillaries and the inspired air.

We are beginning to know something about the clinical history and the relative seriousness of atelectasis of this type. We know, for instance, that it occurs with greatest frequency in infants born prematurely, in infants born of diabetic mothers and in infants born by cesarean section. We know also something about the symptomatology. It has as a rule a rather uniform pattern (there are some exceptions). The babies usually breathe normally at birth, or seem to, and their respirations are well established without any particular difficulty or symptoms of respiratory distress. Symptoms of respiratory distress do not as a rule become very clearly apparent until several hours—maybe three, four or five hours—after birth. However, studies have shown that if the respiratory rate of these infants were being watched and counted constantly, evidence of trouble might be noted very much earlier. According to Dr. Herbert Miller[3] of the University of Kansas, the respiratory rate in most instances definitely tends to increase very shortly after birth and continues to increase until it is abnormally high; and by that time the previously mentioned classic signs are present.

The signs of atelectasis due to hyaline membrane disease differ in some respects from those of atelectasis of other types. In addition to the rapid breathing, there is intercostal retraction and an audible expiratory grunt, and in many cases rather definite retraction of the lower sternum. These signs persist and tend to increase in severity. Cyanosis is likely to be present at some stage as time goes on, sometimes becoming quite severe and relieved only by oxygen administration.

One thing that impresses me more than anything else as I have observed children with atelectasis due to hyaline membrane is the tremendous amount of work they do in order to breathe, and it is a matter of great wonder that they can keep it up hour after hour.

Increasing the rate of respiration and the depth of respiration is necessary in order to get enough air to overcome the dead space and reach the alveoli for sufficient oxygen and exchange. Survival depends largely upon the strength of the infant and his ability to carry this tremendous work-load long enough for the process to spontaneously resolve itself. There is an interesting clinical phenomenon in connection with the labored breathing: If the baby is strong enough to carry this load long enough, then toward the end of the third day or the beginning of the fourth day or about that time, there seems to be a rather abrupt and definite improvement in his clinical condition, although sometimes rapid respirations will continue for a time and often the roentgenographic abnormalities persist a little longer. The sudden resolving of this process is reminiscent of the crisis in the normal course of lobar pneumonia.

X-ray films are of some help in diagnosis. Peterson and Pendleton[4] published an article in the *American Journal of Roentgenology, Radiology, Radiological Therapy* and *Nuclear Medicine* in 1955, describing the roentgenological appearance of atelectasis in hyaline membrane syndrome as being a fine, diffuse, reticulogranular or ground glass pattern in contrast to the coarse and irregular pattern seen in the fetal aspiration syndrome. They also noted that while emphysema, interstitial emphysema or air trapping and pneumothorax and pneumomediastinum are rather commonly seen in the atelectasis of the aspiration type, these conditions are virtually

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not seen at all in atelectasis due to hyaline membrane. Also, in making the diagnosis, all of us appreciate how important it is to know something about the history—history of the pregnancy, of the labor and of the delivery. If a baby is born of a diabetic mother—often such babies are born somewhat before term, after 36 to 37 weeks of gestation, and often by section—we are facing the possibility of a baby that might have hyaline membrane atelectasis. Also with a baby born prematurely we are facing that possibility. Toxemia is another condition in which hyaline membrane is the cause of one type of atelectasis that is troublesome. We often make a diagnosis of hyaline membrane syndrome on the symptoms alone and we have no way of knowing for certain that the atelectasis is due to hyaline membrane except in the case of babies that die and are examined postmortem. However, if the roentgenological symptoms are of the previously mentioned rather uniform pattern, we may possibly be aided in making, with some assurance, a diagnosis that otherwise would be rather speculative.

As far as treatment is concerned, prayer, I think, is helpful. There are some things we can do prophylactically—I mean that if it is possible to prolong gestational age and make the baby who is born prematurely less premature than he might have been, we have done quite a little. If we can avoid doing cesarean section except when it is absolutely necessary, we can do quite a little more. Immediate care of the baby after birth, I think, is quite important —making sure that postural drainage is effected immediately, and that nasopharyngeal suction is applied. If we wish to follow Sidney Gellis'[2] recommendation that the stomach contents be removed to avoid the possibility that vomitus might be drawn into the lungs later, there is certainly no reason not to do so. The baby should be placed in an incubator to keep it warm and left in a posture to aid drainage, with the head slightly below the rest of the body at an incline of about 15 degrees. It is probably best to turn it on its side rather than on its back. We give the baby nothing by mouth for the first 24 hours or, probably preferably, even 48 hours, especially if there are beginning signs of respiratory difficulties. As to active treatment, there are some things that we can do: We can use mist nebulization to keep the atmosphere moist or supersaturated, and if the baby is cyanotic we can give it oxygen. That is probably about as much as can be done.

I would like to end up by saying that, as far as treatment is concerned, I would recommend a large measure of intelligent neglect or what might well be called masterful inactivity.

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Obstetrician's Point of View

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HYALINE MEMBRANE DISEASE has been indicated as the causative agent in from 15 to 50 per cent of neonatal deaths. Approximately one of every 400 newborn dies of this pulmonary syndrome and it is estimated that 25,000 deaths are caused from this condition each year in the United States. In a series of neonatal deaths from 1939 to 1949, Potter[5] found hyaline membrane disease to be the only significant pathological change in 40 per cent of the infants. In a recent communication, Dr. C. A. Smith[8] of Harvard University, said that this condition was the largest single cause of neonatal mortality; the incidence in premature neonatal deaths was about 50 per cent.

The most widely circulated theories as to cause have involved aspiration of amniotic fluid, emphysema and atelectasis, hyperoxia, hormonal influence, infection, metabolic change, protein derived from blood, cardiac failure, vagal nerve dysfunction and a deficiency of the precursors of fibrinolysin.

Most authorities are in agreement that prematurity is the one great single contributing element in neonatal deaths due to this syndrome. Infants of diabetic mothers have also been shown to be especially likely to develop this condition. There is a great deal of controversy as to whether there is any significant increase in the incidence of hyaline membrane disease in infants delivered by cesarean section. Rodgers and Gruenwald[7] are of the opinion that infants born of mothers with antepartum bleeding also have a greater predisposition toward the development of hyaline membrane.

Thus the role of the obstetrician in combating this condition is first and foremost to do all he can to prevent prematurity. In the case of diabetic mothers, complete cooperation with a competent internist and

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